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 NEWS 12  OCT 17   STN(R) AnaVist(TM), Version 1.01, allows the export/download
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 NEWS 14  OCT 27   DIOGENES content streamlined
 NEWS 15  OCT 27   EPFULL enhanced with additional content

 NEWS EXPRESS  JUNE 13 CURRENT WINDOWS VERSION IS V8.0, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
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FILE 'HOME' ENTERED AT 07:27:02 ON 29 OCT 2005

=> file medline, uspatful, dgene, embase, wpids, biosis
COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                 0.21       0.21
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FILE 'MEDLINE' ENTERED AT 07:27:47 ON 29 OCT 2005

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=> s cholecystokinin release
L1 673 CHOLECYSTOKININ RELEASE

=> s l1 and method
L2 62 L1 AND METHOD

=> s l2 and lysine residue
L3 0 L2 AND LYSINE RESIDUE

=> s l2 and oligomeric moiety
L4 0 L2 AND OLIGOMERIC MOIETY

=> s l1 and luminal cholecystokinin
L5 9 L1 AND LUMINAL CHOLECYSTOKININ

=> d l5 ti abs ibib tot

L5 ANSWER 1 OF 9 MEDLINE on STN
TI Inhibitory effect of somatostatin on **cholecystokinin release** is independent of **luminal cholecystokinin**-releasing factor content in conscious rats.
AB INTRODUCTION: Exclusion of bile-pancreatic juice from the intestine increases pancreatic secretion via cholecystokinin (CCK) release in conscious rats. Luminal CCK-releasing factor (LCRF), purified from rat intestinal secretions, is an intraluminal regulator of CCK secretion during bile-pancreatic juice diversion. AIMS: Because somatostatin is a potent inhibitor of CCK release and pancreatic secretion, the inhibitory effect of somatostatin on LCRF was examined. METHODOLOGY: Rats were prepared with bile and pancreatic cannulae and two duodenal cannulae and with an external jugular vein cannula. The experiments were conducted without anesthesia. After 1.5-hour basal collection of pancreatic juice with bile-pancreatic juice return, bile-pancreatic juice was diverted for 2 hours, during which time somatostatin (2, 10 nmol/kg/h) was infused intravenously. The rats were killed before and 1 and 2 hours after bile-pancreatic juice diversion. To examine the effect of luminal somatostatin, 50 or 200 nmol/kg/h of somatostatin was infused into the duodenum. The plasma CCK and luminal content of LCRF were measured by specific radioimmunoassays. RESULTS: Bile-pancreatic juice diversion significantly increased pancreatic secretion, plasma CCK, and LCRF levels. Intravenous infusion of somatostatin inhibited CCK release and pancreatic secretion, but not LCRF content. Luminal administration of somatostatin did not show any effect. CONCLUSION: Inhibitory effect of circulating somatostatin on CCK release and pancreatic secretion is independent of LCRF content.
ACCESSION NUMBER: 2001565358 MEDLINE

DOCUMENT NUMBER: PubMed ID: 11668212
TITLE: Inhibitory effect of somatostatin on **cholecystokinin release** is independent of **luminal cholecystokinin**-releasing factor content in conscious rats.
AUTHOR: Miyasaka K; Masuda M; Kanai S; Ohta M; Suzuki S; Tateishi K; Funakoshi A
CORPORATE SOURCE: Department of Clinical Physiology, Tokyo Metropolitan Institute of Gerontology, 35-2 Sakaecho, Itabashiku, Tokyo-173-0015, Japan.. miyasaka@tmig.or.jp
SOURCE: Pancreas, (2001 Nov) 23 (4) 414-20.
Journal code: 8608542. ISSN: 0885-3177.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011023
Last Updated on STN: 20020124
Entered Medline: 20011231

L5 ANSWER 2 OF 9 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN
TI Inhibitory effect of somatostatin on **cholecystokinin release** is independent of **luminal cholecystokinin**-releasing factor content in conscious rats.
AB Introduction: Exclusion of bile-pancreatic juice from the intestine increases pancreatic secretion via cholecystokinin (CCK) release in conscious rats. Luminal CCK-releasing factor (LCRF), purified from rat intestinal secretions, is an intraluminal regulator of CCK secretion during bile-pancreatic juice diversion. Aims: Because somatostatin is a potent inhibitor of CCK release and pancreatic secretion, the inhibitory effect of somatostatin on LCRF was examined. Methodology: Rats were prepared with bile and pancreatic cannulae and two duodenal cannulae and with an external jugular vein cannula. The experiments were conducted without anesthesia. After 1.5-hour basal collection of pancreatic juice with bile-pancreatic juice return, bile-pancreatic juice was diverted for 2 hours, during which time somatostatin (2, 10 nmol/kg/h) was infused intravenously. The rats were killed before and 1 and 2 hours after bile-pancreatic juice diversion. To examine the effect of luminal somatostatin, 50 or 200 nmol/kg/h of somatostatin was infused into the duodenum. The plasma CCK and luminal content of LCRF were measured by specific radioimmunoassays. Results: Bile-pancreatic juice diversion significantly increased pancreatic secretion, plasma CCK, and LCRF levels. Intravenous infusion of somatostatin inhibited CCK release and pancreatic secretion, but not LCRF content. Luminal administration of somatostatin did not show any effect. Conclusion: Inhibitory effect of circulating somatostatin on CCK release and pancreatic secretion is independent of LCRF content.
ACCESSION NUMBER: 2001373902 EMBASE
TITLE: Inhibitory effect of somatostatin on **cholecystokinin release** is independent of **luminal cholecystokinin**-releasing factor content in conscious rats.
AUTHOR: Miyasaka K.; Masuda M.; Kanai S.; Ohta M.; Suzuki S.; Tateishi K.; Funakoshi A.
CORPORATE SOURCE: Dr. K. Miyasaka, Department of Clinical Physiology, Tokyo Metropol. Inst. of Gerontology, 35-2 Sakaecho, Itabashiku, Tokyo 173-0015, Japan. miyasaka@tmig.or.jp
SOURCE: Pancreas, (2001) Vol. 23, No. 4, pp. 414-420.
Refs: 38
ISSN: 0885-3177 CODEN: PANCE4
COUNTRY: United States

DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
003 Endocrinology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20011108
Last Updated on STN: 20011108

L5 ANSWER 3 OF 9 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Compositions useful in treatment of obesity include **luminal cholecystokinin** releasing factor coupled to an amphiphilic polymer, which exhibits improved pharmacokinetic properties.
AN 2001-496568 [54] WPIDS
AB WO 200141812 A UPAB: 20040210
NOVELTY - Compositions which include **luminal cholecystokinin** releasing factor (LCRF) coupled to amphiphilic polymers are new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) LCRF composition comprising LCRF coupled with one or more molecules of a non-naturally occurring polymer. The polymer comprises a lipophilic group and a hydrophilic polymer group, therefore imparting both lipophilic and hydrophilic characteristics to the composition so that the composition is soluble in pharmaceutical solvents and is able to interact with biological membranes;

(2) peptide composition comprising LCRF coupled with one or more molecules of a non-naturally occurring polymer which comprises a LM and a hydrophilic moiety. The composition is soluble in aqueous solvents and the LCRF is active in treatment or prevention of obesity;

(3) LCRF composition comprising LCRF covalently coupled with one or more molecules of a polymer which comprises a linear polyalkylene glycol group and a lipophilic group. The peptide and components are conformationally arranged such that the LCRF has an enhanced in vivo resistance to enzymatic degradation, relative to LCRF alone;

(4) multiligand conjugated LCRF complex comprising a triglyceride backbone group. The LCRF is covalently coupled with the triglyceride backbone group through a polyalkylene glycol spacer group which is bonded at a carbon atom of the triglyceride backbone. At least one fatty acid is covalently attached to a carbon atom of the triglyceride backbone group or is covalently joined through a polyalkylene glycol spacer group;

(5) stable, aqueous-soluble, conjugated LCRF complex which comprises a LCRF conjugatively coupled to a glycolipid group modified with polyethylene glycol;

(6) polysorbate complex comprising a polysorbate group which includes a triglyceride backbone which has a fatty acid group covalently coupled to one of the alpha , alpha ' or beta carbon atoms and a polyethylene glycol group covalently coupled to one of the alpha , alpha ' or beta carbon atoms. A physiologically active moiety can be covalently bonded to the polyethylene glycol group;

(7) compounds of formula (I):

X = N, O or S;

Y = LCRF or a protein;

n = 3 - 230; and

m = 0 - 20.

ACTIVITY - Anorectic. No biodata is provided.

MECHANISM OF ACTION - **Luminal cholecystokinin** releasing factor receptor agonist.

USE - The materials are useful for delivery of LCRF to receptors in the gut. LCRF is capable of stimulating release of cholecystokinin, a polypeptide hormone that induces satiety and reduces food intake. The materials may thus be used in treatment or prevention of obesity. Other peptides may be used in place of LCRF in the materials, so that they could be used for delivery of peptides useful in treatment of other disorders.

ADVANTAGE - The materials are stable and soluble in aqueous solutions. They may exhibit prolonged blood circulation and can be conformationally arranged so that the LCRF has enhanced in vivo resistance to enzymatic degradation. The conjugates can also deliver LCRF to receptors in the gut without absorption into the bloodstream.

Dwg.0/3

ACCESSION NUMBER: 2001-496568 [54] WPIDS
DOC. NO. CPI: C2001-149074
TITLE: Compositions useful in treatment of obesity include **luminal cholecystokinin** releasing factor coupled to an amphiphilic polymer, which exhibits improved pharmacokinetic properties.
DERWENT CLASS: A25 A96 B04 D16
INVENTOR(S): EKWURIBE, N N; EKWURIBE, N
PATENT ASSIGNEE(S): (NOBE-N) NOBEX CORP; (EKWU-I) EKWURIBE N
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001041812	A2	20010614	(200154)*	EN	49
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001020875	A	20010618	(200161)		
BR 2000016339	A	20020827	(200265)		
NO 2002002793	A	20020813	(200266)		
EP 1237580	A2	20020911	(200267)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
CZ 2002001990	A3	20021113	(200282)		
KR 2002068053	A	20020824	(200309)		
JP 2003516366	W	20030513	(200334)		59
HU 2003000133	A2	20030528	(200341)		
CN 1434725	A	20030806	(200366)		
US 6638906	B1	20031028	(200372)		
MX 2002005885	A1	20021101	(200376)		
ZA 2002004603	A	20031126	(200402)		71
NZ 519489	A	20040130	(200414)		
US 2004092449	A1	20040513	(200432)		
IN 2002000752	P3	20050304	(200547)	EN	

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001041812	A2	WO 2000-US33592	20001211
AU 2001020875	A	AU 2001-20875	20001211
BR 2000016339	A	BR 2000-16339	20001211
		WO 2000-US33592	20001211
NO 2002002793	A	WO 2000-US33592	20001211
		NO 2002-2793	20020612
EP 1237580	A2	EP 2000-984215	20001211
		WO 2000-US33592	20001211
CZ 2002001990	A3	WO 2000-US33592	20001211
		CZ 2002-1990	20001211
KR 2002068053	A	KR 2002-707500	20020612
JP 2003516366	W	WO 2000-US33592	20001211
		JP 2001-543156	20001211
HU 2003000133	A2	WO 2000-US33592	20001211

		HU 2003-133	20001211
CN 1434725	A	CN 2000-818964	20001211
US 6638906	B1	US 1999-459443	19991213
MX 2002005885	A1	WO 2000-US33592	20001211
		MX 2002-5885	20020612
ZA 2002004603	A	ZA 2002-4603	20020607
NZ 519489	A	NZ 2000-519489	20001211
		WO 2000-US33592	20001211
US 2004092449	A1 Div ex	US 1999-459443	19991213
		US 2003-633966	20030804
IN 2002000752	P3	IN 2002-MN752	20020610
		WO 2000-US33592	

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001020875	A Based on	WO 2001041812
BR 2000016339	A Based on	WO 2001041812
EP 1237580	A2 Based on	WO 2001041812
CZ 2002001990	A3 Based on	WO 2001041812
JP 2003516366	W Based on	WO 2001041812
HU 2003000133	A2 Based on	WO 2001041812
MX 2002005885	A1 Based on	WO 2001041812
NZ 519489	A Based on	WO 2001041812
US 2004092449	A1 Div ex	US 6638906

PRIORITY APPLN. INFO: US 1999-459443 19991213; US 2003-633966 20030804

L5 ANSWER 4 OF 9 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

TI New isolated **luminal cholecystokinin**-releasing polypeptide - used to suppress appetite, to stimulate gall bladder emptying, for inhibiting gastric emptying or for stimulating insulin secretion..

AN 1997-259024 [23] WPIDS

AB WO 9715671 A UPAB: 19981021

An isolated cholecystokinin-releasing polypeptide (CRP) is claimed which specifically binds with antibodies raised against a polypeptide having at least the amino acid sequence (I):STFWAYQPDGDNDPTDYQKYEHTSSPSQLLAPGDYPCVIEV (I). Also claimed are: (1) an isolated polypeptide comprising the amino acid sequence (I); (2) an isolated CRP or functional or homologous variants comprising: (a) the amino acid sequence (I); or (b) the amino acid sequence (I) from position 1-35, 11-25, 7-23, or 22-37; or (c) the amino acid sequence (I) from position 1-35 where lysine is replaced with alanine at position 19; (3) a purified antibody that specifically binds to a polypeptide as in (1); (4) an isolated nucleic acid segment (II) that encodes a CRP which specifically binds with antibodies raised against a polypeptide having at least the partial amino acid sequence (I); (5) an isolated nucleic acid (III) segment that encodes a polypeptide comprising the amino acid sequence (I); (6) a recombinant vector comprising (II) or (III); and (7) a recombinant host cell comprising a recombinant vector as in (6).

USE - The CRP polypeptides mediate negative feedback regulation of pancreatic enzyme secretion as well as cholecystokinin (CCK) release. They can be used for the treatment of conditions related to lack of or insufficient regulation of CCK release. They can be used to suppress appetite, for stimulating gallbladder contraction or treating gallbladder disease related to gallstone formation, for inhibiting gastric emptying or for stimulating insulin secretion (claimed). The peptides and antibodies can also be used for detection, purification, inhibition studies and immunolocalisation studies (kits provided).

ADVANTAGE - The CRP polypeptides can be administered orally to mimic

the CCK release that food (particularly fat and protein) causes, but lacking the calories.

Dwg.0/24

ACCESSION NUMBER: 1997-259024 [23] WPIDS
DOC. NO. NON-CPI: N1997-214141
DOC. NO. CPI: C1997-083743
TITLE: New isolated **luminal cholecystokinin** -releasing polypeptide - used to suppress appetite, to stimulate gall bladder emptying, for inhibiting gastric emptying or for stimulating insulin secretion..
DERWENT CLASS: B04 D16 S03
INVENTOR(S): GREEN, G M; KRAIG, E B; LIDDLE, R A; REEVE, J R
PATENT ASSIGNEE(S): (UYDU-N) UNIV DUKE; (TEXA) UNIV TEXAS SYSTEM; (REGC) UNIV CALIFORNIA
COUNTRY COUNT: 74
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9715671	A1	19970501	(199723)*	EN	157
RW: AT BE CH DE DK EA ES FI FR GB GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG UZ VN					
AU 9711179	A	19970515	(199736)		
NO 9801857	A	19980624	(199835)		
EP 862631	A1	19980909	(199840)	EN	
R: AT BE CH DE DK ES FI FR GB IE IT LI NL SE					
AU 708857	B	19990812	(199944)		
NZ 324100	A	19991129	(200031)		
JP 2000515721	W	20001128	(200065)		137
MX 9803314	A1	20000901	(200139)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9715671	A1	WO 1996-US17998	19961023
AU 9711179	A	AU 1997-11179	19961023
NO 9801857	A	WO 1996-US17998	19961023
		NO 1998-1857	19980424
EP 862631	A1	EP 1996-941980	19961023
		WO 1996-US17998	19961023
AU 708857	B	AU 1997-11179	19961023
NZ 324100	A	NZ 1996-324100	19961023
		WO 1996-US17998	19961023
JP 2000515721	W	WO 1996-US17998	19961023
		JP 1997-516871	19961023
MX 9803314	A1	MX 1998-3314	19980427

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9711179	A	Based on	WO 9715671
EP 862631	A1	Based on	WO 9715671
AU 708857	B	Previous Publ.	AU 9711179
		Based on	WO 9715671
JP 2000515721	W	Based on	WO 9715671

PRIORITY APPLN. INFO: US 1995-5872P 19951026

L5 ANSWER 5 OF 9 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Cholecystokinin and cholecystokinin receptors.
ACCESSION NUMBER: 2003:149130 BIOSIS
DOCUMENT NUMBER: PREV200300149130
TITLE: Cholecystokinin and cholecystokinin receptors.
AUTHOR(S): Miyasaka, Kyoko, [Reprint Author]; Funakoshi, Akihiro
CORPORATE SOURCE: Department of Clinical Physiology, Tokyo Metropolitan Institute of Gerontology, 35-2 Sakaecho, Itabashi-ku, Tokyo, 173-0015, Japan
SOURCE: Journal of Gastroenterology, (January 2003) Vol. 38, No. 1, pp. 1-13. print.
ISSN: 0944-1174 (ISSN print).
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
OTHER SOURCE: GenBank-D85606
ENTRY DATE: Entered STN: 19 Mar 2003
Last Updated on STN: 9 May 2003

L5 ANSWER 6 OF 9 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Inhibitory effect of somatostatin on **cholecystokinin release** is independent of **luminal cholecystokinin**-releasing factor content in conscious rats.
AB Introduction: Exclusion of bile-pancreatic juice from the intestine increases pancreatic secretion via cholecystokinin (CCK) release in conscious rats. Luminal CCK-releasing factor (LCRF), purified from rat intestinal secretions, is an intraluminal regulator of CCK secretion during bile-pancreatic juice diversion. Aims: Because somatostatin is a potent inhibitor of CCK release and pancreatic secretion, the inhibitory effect of somatostatin on LCRF was examined. Methodology: Rats were prepared with bile and pancreatic cannulae and two duodenal cannulae and with an external jugular vein cannula. The experiments were conducted without anesthesia. After 1.5-hour basal collection of pancreatic juice with bile-pancreatic juice return, bile-pancreatic juice was diverted for 2 hours, during which time somatostatin (2, 10 nmol/kg/h) was infused intravenously. The rats were killed before and 1 and 2 hours after bile-pancreatic juice diversion. To examine the effect of luminal somatostatin, 50 or 200 nmol/kg/h of somatostatin was infused into the duodenum. The plasma CCK and luminal content of LCRF were measured by specific radioimmunoassays. Results: Bile-pancreatic juice diversion significantly increased pancreatic secretion, plasma CCK, and LCRF levels. Intravenous infusion of somatostatin inhibited CCK release and pancreatic secretion, but not LCRF content. Luminal administration of somatostatin did not show any effect. Conclusion: Inhibitory effect of circulating somatostatin on CCK release and pancreatic secretion is independent of LCRF content.
ACCESSION NUMBER: 2001:540888 BIOSIS
DOCUMENT NUMBER: PREV200100540888
TITLE: Inhibitory effect of somatostatin on **cholecystokinin release** is independent of **luminal cholecystokinin**-releasing factor content in conscious rats.
AUTHOR(S): Miyasaka, Kyoko [Reprint author]; Masuda, Masao; Kanai, Setsuko; Ohta, Minoru; Suzuki, Shinji; Tateishi, Kayoko; Funakoshi, Akihiro
CORPORATE SOURCE: Department of Clinical Physiology, Tokyo Metropolitan Institute of Gerontology, 35-2 Sakaecho, Itabashiku, Tokyo, 173-0015, Japan
miyasaka@tmig.or.jp
SOURCE: Pancreas, (November, 2001) Vol. 23, No. 4, pp. 414-420. print.
CODEN: PANCE4. ISSN: 0885-3177.
DOCUMENT TYPE: Article

LANGUAGE: English
ENTRY DATE: Entered STN: 21 Nov 2001
Last Updated on STN: 25 Feb 2002

L5 ANSWER 7 OF 9 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI **Luminal cholecystokinin** releasing factor (LCRF) stimulates CCK release from intestinal endocrine cells through a calcium influx pathway.
ACCESSION NUMBER: 1999:288259 BIOSIS
DOCUMENT NUMBER: PREV199900288259
TITLE: **Luminal cholecystokinin** releasing factor (LCRF) stimulates CCK release from intestinal endocrine cells through a calcium influx pathway.
AUTHOR(S): Liddle, R. A. [Reprint author]; Prpic, V. [Reprint author]; Wang, Y. [Reprint author]; Romac, J. [Reprint author]; Green, G. M. [Reprint author]; Reeve, J. R. [Reprint author]
CORPORATE SOURCE: Duke Univ Med Ctr, Durham, NC, USA
SOURCE: Gastroenterology, (April, 1999) Vol. 116, No. 4 PART 2, pp. A622. print.
Meeting Info.: Digestive Disease Week and the 100th Annual Meeting of the American Gastroenterological Association. Orlando, Florida, USA. May 16-19, 1999. American Gastroenterological Association.
CODEN: GASTAB. ISSN: 0016-5085.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Aug 1999
Last Updated on STN: 5 Aug 1999

L5 ANSWER 8 OF 9 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Atropine-resistant secretion of a putative luminal CCK-releasing peptide in conscious rats.
AB The changes in levels of the newly discovered luminal CCK-releasing factor (LCRF) in the small intestinal lumen before and after bile-pancreatic juice diversion in conscious rats were examined by a specific RIA. Moreover, we also examined whether LCRF secretion was under cholinergic control. Anti-LCRF antiserum was raised in rabbits, and a sensitive RIA was established. The localization of LCRF was examined by immunohistochemistry. The luminal content of LCRF was significantly increased by bile-pancreatic juice diversion, during which luminal trypsin activity was eliminated. The increase in luminal LCRF content was not inhibited by intravenous infusion of atropine. The changes in plasma levels of CCK and pancreatic secretion were similar to those in luminal LCRF contents. LCRF immunostaining was observed in villus tip enterocytes of the small intestine and was most prominent in the duodenal portion. These results support our original hypothesis that LCRF may be released spontaneously into the small intestinal lumen from the villus tip enterocytes and its intraluminal degradation by proteases regulates CCK release. Furthermore, LCRF release was not subject to cholinergic regulation.
ACCESSION NUMBER: 1999:197321 BIOSIS
DOCUMENT NUMBER: PREV199900197321
TITLE: Atropine-resistant secretion of a putative luminal CCK-releasing peptide in conscious rats.
AUTHOR(S): Miyasaka, Kyoko [Reprint author]; Tateishi, Kayoko; Masuda, Masao; Jimi, Atsuo; Funakoshi, Akihiro
CORPORATE SOURCE: Department Clinical Physiology, Tokyo Metropolitan Institute Gerontology, 35-2 Sakae-cho, Itabashiku, Tokyo 173, Japan
SOURCE: American Journal of Physiology, (Jan., 1999) Vol. 276, No. 1 PART 1, pp. G287-G292. print.

CODEN: AJPHAP. ISSN: 0002-9513.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 25 May 1999
Last Updated on STN: 25 May 1999

L5 ANSWER 9 OF 9 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Luminal feedback regulation, monitor peptide, CCK-releasing peptide, and CCK receptors.
AB We summarize the discovery of luminal feedback regulation of pancreatic secretion in rats and its history. In rats, removal of proteolytic activity from the intestine produced a significant increase in pancreatic protein (enzyme) output. This increase was confirmed to be mediated by circulating cholecystokinin (CCK). Subsequently, two CCK-releasing peptides, monitor peptide and luminal CCK-releasing factor (LCRF), were purified from the rat pancreatic juice and small intestine, respectively, to elicit CCK release in luminal feedback regulation. Furthermore, we emphasize the important physiologic roles of CCK and CCK receptors by the discovery of disrupted CCK-A-receptor gene in rats. These findings should help to determine the regulation of pancreatic secretion and CCK functions in humans.
ACCESSION NUMBER: 1998:209668 BIOSIS
DOCUMENT NUMBER: PREV199800209668
TITLE: Luminal feedback regulation, monitor peptide, CCK-releasing peptide, and CCK receptors.
AUTHOR(S): Miyasaka, Kyoko [Reprint author]; Funakoshi, Akihiro
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=> s l1 and branched moiety
L6            0 L1 AND BRANCHED MOIETY

=> d his

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     FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, BIOSIS' ENTERED AT
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L1          673 S CHOLECYSTOKININ RELEASE
L2           62 S L1 AND METHOD
L3            0 S L2 AND LYSINE RESIDUE
L4            0 S L2 AND OLIGOMERIC MOIETY
L5            9 S L1 AND LUMINAL CHOLECYSTOKININ
L6            0 S L1 AND BRANCHED MOIETY

=> s l1 and hydrolyzable linker
L7            0 L1 AND HYDROLYZABLE LINKER
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